A study on non diabetic soft tissue infections

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BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr. K.A.Sivasubramanian on "a study on non diabetic soft tissue infections"**during his M.S. (General Surgery) course from May 2007 to March 2010 at the Government Kilpauk Medical College and Government Royapettah Hospital, Chennai .

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TABLE OF CONTENTS

INTRODUCTION

Soft tissue infections are a diverse group of diseases that involve the skin and underlying subcutaneous tissue, fascia, or muscle. Such infections may be localized to a small area or may involve a large portion of the body. They may affect any part of the body, though the lower extremities, the perineum, and the abdominal wall are the most common sites of involvement.

Soft tissue infections can be localised or spreading and can be necrotising or non necrotising .localised or non spreading non necrotising infections usually respond to broad spectrum antibiotics. Localised necrotising fascitis will respond to surgical debridement and antibiotics. Spreading necrotising soft tissue infections constitute a life threatening surgical emergency, requiring immediate resuscitation, intravenous antibiotics and urgent surgical intervention with radical debridement.

Soft tissue infections who presents with abscess, cellulitis, necrotising fascitis, gas gangrene are common among the diabetes due to their raised blood sugar, neuropathy which leads to unnoticed infections, vascular insufficiency due to angiopathy.

There is also a substantial raise in the incidence of infection among the non diabetics in my unit .hence a study on non diabetic soft tissue infections where made in my unit during the period of may 2007 to November 2009.

This is a Prospective study involving 50 cases and a detailed study was made to sort out their aetiology, mode of presentation, most frequent organisms and their outcome.

HISTORICAL REVIEW

Soft tissue infections were first defined as such slightly more than a century ago.

In 1883, Fournier described a gangrenous infection of the scrotum that continues to be associated with his name.

In 1924, Meleney documented the pathogenic role of streptococci in soft tissue infection. shortly thereafter, brewer and Meleney described progressive polymicrobial postoperative infection of the muscular fascia with necrosis (though the term necrotizing fasciitis was not introduced until more than 25 years later.

The association between toxic-shock syndrome and streptococcal soft tissue infection was delineated as this disease re-emerged in the 1980s

AIM OF THE STUDY

1. TO STUDY THE PROBABLE ETIOLOGY.

2. TO STUDY THE AGE DISTRIBUTION AMONG THE PATIENTS.

3. THE MODE OF PRESENTATION.

4. THE COMMONEST SITE AND THE ORGANISMS AND ITS SENSITIVITY.

5. UNDERLYING OTHER DISORDERS.

6. PERCENTAGE OF COMPLICATIONS.

MATERIALS & METHODS

TYPE OF STUDY - PROSPECTIVE STUDY

NUMBER OF CASES - 50 CASES

PERIOD OF STUDY - MAY 2007 TO NOVEMBER 2009

INSTITUTION - DEPARTMENT OF GENERAL SURGERY, GOVERNMENT ROYAPETTAH HOSPITAL, ATTACHED TO KILPAUK MEDICAL COLLEGE

TYPE OF ANALYSIS - CLINICAL DATA ANALYSIS

INCLUSION CRITERIA - SOFT TISSUE INFECTIONS OF THE BODY

EXCLUSION CRITERIA - DIABETIC

- POSTOPERATIVE WOUND INFECTIONS
- SKIN INFECTIONS (FOLLICULITIS,
- FURUNCULOSIS, IMPETIGO)
- ANIMAL & HUMAN BITES
- PERIANAL ABSCESS

LIMITATIONS:

Since most of the patients where taken up for emergency procedures and where relieved of their symptoms, and where discharged earlier a long term follow up could not be made out.

To consolidate our study I excluded perianal abscess since it formed a large proportion of cases in my unit than any other cases of non diabetic soft tissue infections and studies were being made on the perianal sepsis extensively.

MANAGEMENT PROTOCOL:

1. CLINICAL EVALUATION
2. INVESTIGATIONS

    - BLOOD INVESTIGATIONS

        HB%,

        STS/RVT

    - WOUND C/S,

    - X- RAY OF THE LOCAL PART

3. NUTRITION AND IMMUNO DEFICIENCY STATES
4. MANAGEMENT

    CONSERVATIVE (ANTIBIOTICS)

    SURGICAL –I&D,

        FASCIOTOMY,

        SLOUGH EXCISION.

        AMPUTATION

5. POSTOPERTIAVE COMPICATIONS

LITERATURE REVIEW

Skin and Subcutaneous Tissue:

Introduction:

The skin is the largest and among the most complex organs of the body. Its uniform appearance belies its great variation from region to region of the body and the complex organization and interaction of the many different cells and matrices of the skin. Although the skin functions simply as a protective barrier to interface with our environment, its structure and physiology are complex.

In its role as an environmental buffer, the skin protects against most noxious agents, such as chemicals (by the impermeability of the epidermis), solar radiation (by means of pigmentation), infectious agents (through efficient immune-surveillance), and physically deforming forces (by the durability of the dermis). Its efficient ability to conserve or disperse heat makes the skin the major organ responsible for thermoregulation. To direct all these functions, the skin has a highly specialized nervous structure.

These various functions are better served by different components of skin, so that teleologically regional variation develops. The palms and soles are particularly thick, to bear weight. The fingertips have the highest density of sensory innervation and allow for intricate tasks. Even the lines of the skin, first described by Langer, are oriented perpendicularly to the long axis of muscles to allow the greatest degree of stretching and contraction without deformity.

Anatomy and Physiology:

Traditionally the skin has been divided into three layers: the epidermis, the basement membrane, and the dermis .The epidermis is composed mainly of cells, with very little extracellular matrix. Each cell type serves a specific barrier function. Keratinocytes provide a mechanical barrier, melanocytes provide a barrier to radiation, and Langerhans' cells provide an immunologic barrier. The dermis contains mostly extracellular matrix, providing support for nerves, vasculature, and adnexal structures. The dermis allows skin to resist deforming forces and return to its resting state, thus providing durability. The basement membrane is a specialized structure that anchors the epidermis to the dermis.

SKIN IS EXPOSED TO VARIETY OF INSULTS LIKE:

- TRAUMATIC INJURIES
- CAUSTICINJURIES
- PRESSURE SORES
- THERMAL INJURIES
- RADIATION INJURIES
- INFECTIOUS DISEASES
- INFLAMMATORY DISORDERS
- TUMORS

Here is a detailed discussion about the infectious diseases of skin. The commonest diseases affecting the skin, subcutaneous, fascias and muscles are being discussed here.

PLATE I

CELLULITIS_

CELLULITIS RIGHT ARM AND RIGHT LEG

_

CELLULITIS LEFT HAND CELLULITIS RIGHT LEG

ABSCESS AND CELLULITIS

Skin **abscesses** are collections of pus within the dermis and deeper skin tissues.

A **furuncle (or "boil")** is an infection of the hair follicle in which purulent material extends through the dermis into the subcutaneous tissue, where a small abscess forms.

A **carbuncle** is a coalescence of several inflamed follicles into a single inflammatory mass with purulent drainage from multiple follicles.

Etiology;

Skin abscesses, furuncles and carbuncles can develop in healthy individuals with no predisposing conditions other than skin or nasal carriage of **Staphylococcus aureus**; spontaneous infection due to **community-acquired methicillin-resistant S. aureus (CA-MRSA)** may occur with greater frequency than abscesses due to other pathogens.

Superficial abscesses on the trunk and on the **head and neck** are most commonly caused by ***S. aureus***, often combined with **streptococci.**

Abscesses in the axillae frequently have a prominent gram-negative component.

The highest rates of anaerobes in wounds were in the inguinal, buttocks, and trunk areas and in abscesses in the perirectal, external genitalia, neck, and inguinal areas.). The predominant anaerobic organisms were **Bacteroides** species, **Peptostreptococcus** species, **Clostridium** species, and Fusobacterium species.(Arch Surg 1990 Nov;125(11):1445-51)

Abscesses **below the waist**, especially on the perineum, are often found to harbour **mixed aerobic and anaerobic gram-negative flora.**

Most abscesses are caused by infection. However, sterile abscesses can occur in the setting of injected irritants. Examples include injected drugs (particularly oil based ones) which may not be fully absorbed so remain at the site of injection, causing local irritation. Sterile abscesses can turn into hard, solid lesions as they scar.

It is recognized clinically as a localized swelling with signs of inflammation and tenderness.

PLATE II

ABSCESS WITH CELLULITIS

CELLULTIS LEFT LEG **ABSCESS RIGHT HAND**

TRUMATIC CELLULITIS RIGHT ARM **POST INFECTIVE RAW AREA**

Treatment;

An abscess will not resolve unless the pus is drained and evacuated.

It is suggested that free drainage following incision and drainage is the safest treatment for the majority of abscesses. Antibiotics do not have any significant effect on healing time or recurrence and their routine use is not recommended.

(Br J Surg 1977 Apr;64(4):264-6.)

An abscess must be distinguished from cellulitis.

Cellulitis

Cellulitis is an acute bacterial infection of the dermis and the subcutaneous tissue with an intact blood supply and viable tissue that is marked by an acute inflammatory response with small vessel engorgement and stasis, endothelial leakage with interstitial oedema, and polymorphonuclear leukocyte infiltration; it is typically located in a more superficial plane that primarily affects the lower extremities, though it can affect other areas as well (e.g., the periorbital, buccal, and perianal regions; the areas around incisions).

Cellulitis resolves with appropriate antibiotic therapy alone if treatment is initiated before tissue death occurs.

An abscess may be mistaken for cellulitis when the central necrotic portion is located deep beneath overlying tissue layers and it cannot be readily detected by physical examination.

It may also be disguised in anatomic locations where fibrous septa join skin and fascia and divide subcutaneous tissue into compartments that limit the local expression of fluctuance while leading to high pressure that causes ischemia and promotes early tissue death. Examples of

such infections include perirectal abscesses, breast abscesses, carbuncles on the posterior of the neck and upper part of the back, and infections in the distal phalanx of the finger (felon).

TRAUMATIC WOUNDS:

Any process leading to a breach in the skin barrier can also predispose to the development of a skin abscesses, furuncle or carbuncle. Examples include primary dermatologic conditions as well as trauma related to abrasions, shaving and insect bites.

Traumatic wounds, when closed and infected, become a surgical complication that needs to be opened, drained, and treated with antibiotics if associated with cellulitis or systemic compromise.

Wounds older than 6 hours, those with significant contamination (dirty, including human and animal bites), wounds associated with necrotic or ischemic tissue, puncture wounds, those classified as stab wounds or gunshot wounds, and wounds caused by a significant crush mechanism or avulsion are not closed. These wounds, as well as those deeper than 1 cm and wounds caused by burns or a frostbite mechanism, receive tetanus prophylaxis if the most recent tetanus booster occurred 5 or more years earlier. The use of antibiotics for simple extremity lacerations has not been proved to reduce the risk for infection after closure

PLATE III

NECROTISING FASCITIS

NECROTISING FASCITIS OF LEFT FOREARM AND RIGHT LEG

FOURNIERS GANGRENE

Necrotizing Soft Tissue Infections

Necrotizing infections of the skin and fascia include necrotizing forms of cellulitis and fasciitis types I and II.

These infections are characterized clinically by fulminant destruction of tissue, systemic signs of toxicity, and a high rate of mortality.

Common **pathologic features** are extensive tissue destruction, thrombosis of blood vessels, abundant bacteria spreading along fascial planes, and an unimpressive infiltration of acute inflammatory cells. Accurate diagnosis and appropriate treatment must include early surgical intervention.

(NSTIs) are less common are much more serious conditions whose severity may initially be unrecognized They typically involve deep subcutaneous tissue, superficial or deep fascia, or muscle, or any combination of

the three NSTIs are characterized by the absence of clear local boundaries or palpable limits. This lack of clear boundaries accounts for both the severity of the infection and the frequent delay in recognizing its surgical nature.

Anatomically, these infections are marked by a layer of necrotic tissue that is not walled off by a surrounding inflammatory reaction and thus is not typically manifested as an abscess unless it is the initiating factor. In addition, the overlying skin has a relatively normal appearance in the early stages of infection, and the visible degree of involvement is substantially less than that of the underlying tissues.

NSTIs have been described by a variety of different labels, including ***gas gangrene*** **and** ***necrotizing fasciitis.*** A substantial number of classifications based on anatomic location, microbiology, and depth of infection, among others, have also been described. The wide range of classifications makes understanding of this entity rather confusing, when the only important factor to be determined is the presence or absence of a necrotic component requiring surgical intervention. If suspected, the approach to diagnosis and management of all patients is the same, thus making detailed classification schemes even less useful. We encourage applying the term NSTI to all infections that fit this category

In advanced stages of the disease, patients usually have overt signs of systemic compromise and septic physiology.

Local findings include tense and tender soft tissues associated with ecchymoses or blistering of the skin, or both. The presence of gas detected either by physical examination (crepitus) or by radiographs has been recognized as a grave finding and can be associated with virtually any bacteria, as opposed to the common perception of its unique association with clostridial infections. Most bacteria, especially facultative gram-negative rods such as *E. coli,* make insoluble gases whenever they are forced to use anaerobic metabolism. Thus, the presence of gas in a soft tissue infection implies anaerobic metabolism. Because human tissue cannot survive in an anaerobic environment, gas associated with infection implies dead tissue and therefore a surgical infection. At this stage the disease advances rapidly and must be diagnosed and treated as soon as possible; however, progression from cellulitis or abscess to NSTI may take several days, which can confuse the clinical picture and delay diagnosis

In patients with chronic infections, obese patients, and those at the early stage of the disease, making the diagnosis of NSTI is not straightforward. Different strategies to allow earlier diagnosis and surgical débridement have been proposed. Frozen biopsy and imaging with either computed tomography (CT) or magnetic resonance imaging may be helpful in these scenarios. [42] [43] [44] [45] However, imaging studies, though sensitive, are nonspecific, and if one has

sufficient suspicion to perform a biopsy, the evidence is usually clear to the eye when the incision for biopsy is made.

DIAGNOSIS:

Wall and colleagues[42] described the association of NSTI with **leukocytosis greater than 15,400/mL**^{3} together with a serum sodium level less than 135 mEq/L, and more recently, Wong and associates[43] created a score (laboratory risk indicator for necrotizing fasciitis [LRINEC]) to distinguish NSTI from non-necrotizing soft tissue infections by using the following variables: C-reactive protein, leukocytosis, hemoglobin, sodium, creatinine, and glucose levels.

Use of these scores can certainly help guide the management of patients with suspected NSTI. However, whenever in doubt, an incision over the compromised area for exploration of the site in the OR is mandatory. Typical intraoperative findings consistent with **NSTI include a dishwater-like exudate, dusky tissues, thrombosed vessels, and lack of clear boundaries** allowing finger dissection to spread through the compromised plane abnormally easily.

Frozen section tissue biopsy is a useful adjunct in establishing an early, accurate diagnosis of infectious gangrene.

ORGANISMS:

NSTIs are typically polymicrobial in nature

Staphylococcal and streptococcal species are relatively common causative organisms in combination with anaerobes. ***Vibrio*** **and fungal (mucormycosis**) pathogens have also been described as causing NSTI.

Clostridial infections are worth special mention. They are typically monomicrobial, although they can be seen in combination with other bacteria.

They are often characterized by infection and necrosis of muscles (myonecrosis) and are associated with a significantly worse prognosis

Clostridial infections are more common in patients with intravenous drug use and are accompanied by a very high white blood cell count. These infections require very expeditious and repeated débridement together with supportive care in the intensive care unit (ICU).

The most common organisms associated with clostridial infections are ***Clostridium perfringens, Clostridium novyi,*** **and** ***Clostridium septicum.*** The only other bacteria commonly reported as the sole cause of nonclostridial NSTI is **β-hemolytic** ***Streptococcus pyogenes.***

Comparison of Clostridial and Nonclostridial Infections

	CLOSTRIDIAL MYONECROSIS	NONCLOSTRIDIAL NECROTIZING INFECTIONS
Erythema	Usually absent	Present, often mild
Swelling/edema	Mild to moderate	Moderate to severe
Exudate	Thin	“Dishwater” to purulent
White cells	Usually absent	Present
Bacteria	GPR ± others	Mixed ± GPR
		May be GPC alone
Advanced signs	Hypesthesia	Hypoesthesia
	Bronze discoloration	Ecchymoses
	Hemorrhagic bullae	Bullae
	Dermal gangrene	Dermal gangrene
	Crepitus	± Crepitus
Deep involvement	Muscle > skin	Subcutaneoustissue ± fascia ± muscle(uncommon) > skin
Histology	Minimal inflammation	Acute inflammation
	Muscle necrosis	Microabscesses
		Viable muscle
Physiology	Rapid onset of tachycardia, hypotension, volume deficit, ± intravascular hemolysis	Variable to minimal tachycardia, hypotension, and volume deficit
Treatment		
General	Aggressive cardiopulmonary resuscitation	Aggressive cardiopulmonary resuscitation
Antibiotics	Penicillin G plus broad-spectrum antibiotic	Third-generation cephalosporin or fluoroquinolone plus antianaerobic agent
	Clindamycin may be useful for inhibiting toxin production	Clindamycin may be useful for inhibiting toxin production
Hyperbaric O_2	If it does not delay other treatment	No

	CLOSTRIDIAL MYONECROSIS	NONCLOSTRIDIAL NECROTIZING INFECTIONS
Surgery	Aggressive removal of infected tissue; amputation of extremity often required	Débridement and exposure; not much removal required; usually no amputation
Antitoxin	No	No

TREATMENT

Treatment of NSTI always includes débridement, and additional support is provided by broad-spectrum antibiotics, monitoring, and systemic support. Débridement of all necrotic tissue must be done promptly, with *scheduled* repeat débridement every 24 hours or sooner if indicated by clinical deterioration.

Major amputations may be required to achieve appropriate source control or when the infection spreads to involve the entirety of a limb.

Antibiotic choices include agents with broad activity against facultative gram-negative rods, gram-positive cocci, and anaerobes.

If MRSA is thought to probably be a pathogen in the patient being treated, an antibiotic that covers this organism needs to be added.

Empirical treatments include triple (penicillin, clindamycin, and aminoglycoside/quinolone) or quadruple (plus vancomycin) antibiotic regimens.

When starting empirical antibiotic treatment, combinations that include the use of high-dose clindamycin is strongly considered. Clindamycin has been shown to block exotoxin production from bacteria, one of the key bacterial factors that perpetuates and leads to spreading of the infection through tissues. Narrower antibiotic regimens can be given once a definitive culture with specific sensitivity.

Appropriate single agents include imipenem/cilastatin, meropenem, ertapenem, tigecycline, and piperacillin/tazobactam. .

Preadmission treatment with antibiotics modified the initial clinical picture and often masked the severity of the underlying infection. Polymicrobial synergistic infection was the most common cause with streptococci and enterobacteriaceae being the most common isolates. Group-A sstreptococcus was the most common cause of monomicrobial necrotizing fasciitis. The most common associated comorbidity was diabetes mellitus. Advanced age, two or more associated comorbidities, and a delay in surgery of more than twenty-four hours adversely affected the outcome. Multivariate analysis showed that only a delay in surgery of more than twenty-four hours was correlated with increased mortality ($p<0.05$; relative risk = 9.4).

GAS GANGRENE

PROGNOSIS:

The mortality associated with NSTI has been in the range of 16% to 45%. Multiple prognostic factors have been identified, including the presence of clostridial infection.

Prognostic Score to Predict Mortality in Patients With Necrotizing Soft Tissue Infection at the Time of First Assessment

VARIABLE (ON ADMISSION)	NO. OF POINTS
Heart rate >110 beats/min	1
Temperature <36°C	1
Creatinine >1.5 mg/dL	1
Age >50 yr	3
White blood cell count >40,000	3
Hematocrit >50	3

GROUP CATEGORIES	NO. OF POINTS	MORTALITY RISK
1	0-2	6%
2	3-5	24%
3	≥6	88%

Early operative debridement was demonstrated to reduce mortality among patients with this condition. A high index of suspicion is important in view of the paucity of specific cutaneous findings early in the course of the disease.

Myonecrosis

Myonecrosis (also called necrotizing myositis) is an uncommon infection of muscle, which develops rapidly and is life-threatening. Early recognition and aggressive treatment are essential. The vast majority of infections resulting in necrosis of muscle are due to Clostridium species (gas gangrene) and group A streptococcus (Streptococcus pyogenes). These infections typically evolve from either contiguous spread from an area of trauma or surgery or spontaneous spread from hematogenous seeding of muscle.

Several other clinical entities can be confused with myonecrosis:

- **Staphylococcus aureus** and, less often other organisms, can cause a primary muscle abscess (**pyomyositis)** in the absence of an apparent site of infection. Pyomyositis is more common in tropical areas, in HIV patients, and in children infected with community-associated methicillin-resistant S. aureus (CA-MRSA).
- Necrotizing infections due to **Vibrio vulnificus** can involve the skin, fascia, and muscle and are most common among patients with cirrhosis, consumers of raw seafood, or inhabitants of coastal regions.
- A number of viral infections, such as **acute influenza type A**, can also produce skeletal muscle injury resulting in **rhabdomyolysis.**

Meleney's ulcer

Meleney's ulcer is characterized by central necrosis, erythema, and edema.

In the 1920s, Meleney demonstrated that injection of animals with pathogens isolated from patients with infectious gangrene reproduced the characteristics of infection.[2] Decades later, the importance of bacterial synergy and exotoxins was demonstrated in experiments performed by Seal and Kingston,[42] who showed that a spreading infection developed in 12% of animals that received an intradermal injection of group A Î²-hemolytic streptococci. When S. aureus was coinjected with Î²-hemolytic streptococci, spreading infections developed in 50% of animals, and when the Î±-lysin of S. aureus was coinjected with streptococci, spreading infections developed in 75%.

Streptococcal Toxic-Shock Syndrome;

Hemolytic streptococci were originally described by Meleney as the cause of a synergistic gangrene.

Streptococcal toxic-shock syndrome (STSS) is defined as the isolation of group A streptococci from a normally sterile body site in conjunction with hypotension and either renal impairment, acute respiratory distress syndrome,

abnormal hepatic function, coagulopathy, extensive tissue necrosis, or an erythematous rash.[35] STSS is considered the probable diagnosis when these abnormalities occur in conjunction with isolation of group A streptococci from nonsterile body sites. More than 60% of patients with STSS have bacteremia.

Population-based studies in North America and Europe documented a nearly fivefold increase in group A streptococcal infections between the late 1980s and 1995.[44] The current incidence of group A streptococcal infections in the population of Ontario, Canada, is estimated to be 1.5 per 100,000.[44] STSS develops in approximately 10% to 15% of these patients, necrotizing fasciitis in about 6%.[45] It is likely that the rise in serious group A streptococcal infections reflects an antigenic shift that has increased the virulence of these organisms.

Soft tissue infections associated with STSS typically involve an extremity.

Approximately 70% of patients will progress to necrotizing fasciitis or myositis and will require operative treatment. Only about 50% of patients with streptococcal soft tissue infections have a demonstrable portal of entry for bacteria.

Severe pain is the most common initial symptom of STSS. It is of sudden onset and generally precedes tenderness or other physical findings. Fever is another common early sign.

About 80% of STSS patients show clinical signs of soft tissue infection (e.g., localized swelling, erythema, and tenderness) In approximately 50%, blood pressure is initially normal, but hypotension invariably develops within 4 to 8 hours after presentation.

Hemoglobinuria and an elevated serum creatinine concentration are hallmarks of renal involvement.

Even when adequate resuscitation is provided and antibiotics and vasopressors are given, hypotension persists in the overwhelming majority of patients.

Renal dysfunction can persist or progress for 48 to 72 hours despite treatment. Hypoalbuminemia and hypocalcemia are common. Mild leukocytosis is present initially; however, the percentage of immature neutrophils is generally 40% or higher.

S. pyogenes can be classified into more than 80 different strains on the basis of the M proteins expressed.

M proteins impede phagocytosis of streptococci and induce vascular leakage by forming complexes with fibrinogen. M1 and M3 are associated with the majority of streptococcal necrotizing soft tissue infections.

Streptococcal pyrogenic exotoxins (SPEs) are produced by most streptococci that cause severe soft tissue infection and can be transmitted by bacteriophages to different M types.

They are the cause of the fever, shock, and tissue injury associated with these infections. SPE-A and SPE-B induce the synthesis of tumor necrosis factor-Î (TNF-Î), interleukin-1Î² (IL-1Î²), and IL-6. Peptidoglycan, lipoteichoic acid, and killed organisms also are capable of inducing TNF-Î production.

It has been proposed that M proteins or SPEs act as superantigens. These exotoxins, along with certain staphylococcal toxins (e.g., toxic-shock syndrome toxin-1 [TSST-1] and staphylococcal enterotoxins), can stimulate T cell responses through conventional antigen-presenting cells, as well as through direct binding to the VÎ² region of the T cell receptor. Conventional T cell activation through antigen-presenting cells is a multistage process that stimulates a relatively small percentage of T cells and limits the magnitude of the resultant cytokine response.

The superantigen processing pathway can stimulate more than a thousand times more T cells than the conventional antigen pathway and thus can trigger a massive release of cytokines.

Although S. pyogenes is susceptible to penicillin and other Î²-lactam antibiotics in vitro, clinical treatment failure sometimes occurs when penicillin is used alone.

Clindamycin is more effective than Î²-lactam agents in managing experimental and clinical infections caused by group A streptococci, particularly when necrosis is present.[48] Clindamycin inhibits protein synthesis, and its efficacy is unaffected by inoculum size or the stage of bacterial growth. In particular, it suppresses bacterial toxin synthesis and inhibits M-protein synthesis, thus facilitating phagocytosis of S. pyogenes. Clindamycin also suppresses synthesis of penicillin-binding proteins, and it can act synergistically with penicillin.

Fournier's Gangrene

Fournier's gangrene is a necrotizing fasciitis of the male genitalia and perineum that primarily involves subcutaneous tissues.

The disease can rapidly progress and, although mortality is dependent on severity of disease, it has exceeded 50% in some series.

The most common cause of Fournier's gangrene is infection of the colon, rectum, or lower genitourinary tract or cutaneous infection of the genitals, perineum, or anus. The most common risk factors are diabetes mellitus, alcohol use, and immunocompromised states.

Infections can spread along the dartos, Collie's, and Scarpa's fascia because these fascial planes are continuous. The spread of infection rarely involves the deep fascial planes and musculature.

ORGANISMS:

Both aerobic and anaerobic organisms can cause the infection. The most common isolated organism is ***Escherichia coli;*** other commonly cultured organisms include enterococci, staphylococci, streptococci, *Bacteroides fragilis,* and *Pseudomonas aeruginosa*

CLINICALLY:

The presenting sign is usually a painful swelling and induration of the penis, scrotum, or perineum. Cellulitis, eschar, necrosis, crepitus, foul odor, and fever may be accompanying signs.

TREATMENT:

Aggressive surgical débridement of all necrotic, ischemic, and infected tissue, along with copious irrigation, are critical. Infected tissue is cultured and initial broad-spectrum IV antibiotic coverage (e.g., ampicillin, gentamicin, and clindamycin) instituted. Suprapubic catheter placement can help divert the urine and decrease the risk for further bacterial seeding of the wound.

Patients with FG need extensive debridement and cystostomy or colostomy when necessary. Broad-spectrum triple antimicrobial regimen and aggressive debridement are mandatory. Topical application of unprocessed honey is beneficial to the healing process. A minority of patients require split-thickness skin grafts on denuded areas.

SUPERFICIAL SKIN INFECTIONS:

Erysipelas:

Erysipelas is an acute bacterial infection that principally involves the dermis.

It is almost invariably caused by **S. pyogenes.**

Infection extends through the dermal lymphatic vessels and is typically manifested by a tender, pruritic, intensely erythematous, sharply demarcated, and raised plaque.

Patients complain of pain, often in conjunction with high fever, increased skin warmth, and leukocytosis. Lymphangitis and lymphadenopathy are sometimes present as well. The leg is the most common site of involvement, but erysipelas may also occur on the face, the arms, and the upper thighs.

The factors predisposing to the development of erysipelas of the extremity include local conditions such as tinea pedis (athlete's foot), leg ulcers, and venous stasis dermatitis.[23] Erysipelas tends to be more common in the presence of associated conditions such as lymphedema, diabetes mellitus, alcoholism, immunocompromise, and obesity,[21,23] and it is more likely to recur in patients with these associated diseases and in those whose underlying skin conditions are inadequately treated.

The standard antibiotic treatment for uncomplicated erysipelas is **penicillin**, which is effective in at least 80% of cases. Oral and intravenous antibiotic regimens are equally efficacious. Amoxicillin appears to work as well as penicillin. Patients with erysipelas of the lower extremity should be placed on bed rest, and the involved leg should be elevated to reduce edema and pain. Once the patient is able to resume normal activities, he or she should be fitted with elastic stockings, which help reduce the recurrence of edema and lower the risk of lymphedema. For patients with tinea pedis, a topical antifungal agent is used to treat the infection and prevent recurrence.

Folliculitis;

Folliculitis is an infection of the hair follicle that is typically caused by S. aureus. It is characterized by a painful, tender, erythematous papule with a central pustule. A shaft of hair is often seen in the center of the pustule.

In rare cases, folliculitis is caused by pathogens other than S. aureus, such as Pseudomonas aeruginosa, Klebsiella species, Enterobacter species.

In most patients, folliculitis resolves spontaneously within 7 to 10 days.[22] Topical therapy with clindamycin, erythromycin, or mupirocin ointments or benzoyl peroxide in combination with warm soaks may accelerate resolution. Isotretinoin can be used to treat gram-negative folliculitis.

In patients with refractory or disseminated follicular infections, oral antibiotic therapy is indicated.

Dicloxacillin, erythromycin, cephalexin, cefadroxil, or clindamycin may be given; oral ciprofloxacin is indicated for the treatment of gram-negative folliculitis .Elimination of predisposing factors is important for reducing the likelihood of recurrence.

Furuncles and carbuncles;

Furuncles and carbuncles are deeper infections of the hair follicle that extend beyond the hair follicle to involve the subcutaneous tissue.

For both, S. aureus is the usual causative organism.

A furuncle, or boil, is a small abscess, manifested as a firm, tender, erythematous nodule that tends to occur in skin areas exposed to friction (e.g., the inner thighs and the axilla). Furuncles also may occur on the face, the neck, the upper back, and the buttocks.

Possible predisposing factors include increased friction and perspiration (as seen in obese individuals or athletes), corticosteroid use, diabetes mellitus, and inherited or acquired defects in neutrophil function.

Initial treatment consists of applying warm compresses to help promote drainage and administering an oral antimicrobial agent that is effective against S. aureus (e.g., dicloxacillin, cephalexin, cefadroxil, erythromycin, or clindamycin) With time, the furuncle becomes fluctuant, and the pus coalesces at the skin surface. An incision-and-drainage procedure is necessary when these lesions do not drain spontaneously. This procedure should be performed with local anesthesia, and care should be taken to open the abscess cavity completely. Lesions that have drained spontaneously should be examined to confirm that the

cavity has been opened sufficiently. Failure to drain these lesions adequately may result in recurrence, as well as in progression to a more serious infection.

A **carbuncle** is a deep cutaneous infection involving multiple hair follicles that is characterized by destruction of fibrous tissue septa and consequent formation of a series of interconnected abscesses.

It is typically manifested by a painful, red, tender, indurated area of skin with multiple sinus tracts. Systemic manifestations (e.g., fever and malaise) are common.

Carbuncles occur most frequently on the nape of the neck, the upper part of the back, or the posterior thigh. The thickness of the overlying skin in these areas leads to lateral extension of the infection and loculation. Patients commonly present with relatively large skin lesions that represent a confluence of inflammatory nodules. These lesions are associated with chronic drainage, sinus tracts, and scarring.

An incision-and-drainage procedure is recommended when a fluctuant carbuncle is present. A thorough search for loculated areas should be undertaken to facilitate drainage of deeper accumulations of pus and to ensure adequate treatment. Wide local excision of the involved skin and subcutaneous fat is often necessary to prevent recurrent disease. An oral antistaphylococcal agent

should be given. All patients with hair follicle infections should cleanse the site with chlorhexidine or an iodine-containing solution.

Infections Developing in Damaged Skin;

Infections from bites, from humans or animals, are typically associated with several bacteria. Although Streptococcus and Staphylococcus can be involved (driven in from the skin by a tooth), other organisms common to the mouth may be seen and typically require other or additional antibiotics. Eikenella corrodens is often seen with human bite injuries and Pasteurella multocida is seen with dog and especially cat bite wounds.

The risk of infection depends on the type of bite, the site of injury, the time elapsed from the bite until presentation, host factors, and the management of the wound.

Risk Factors for Soft Tissue Infection Complicating Animal or Human Bite

Location on the hand or the foot

or over a major joint

Location on the scalp or the face

of an infant

Puncture wound

Delay in treatment lasting longer than 12

hr

Immunosuppression

Chronic alcoholism

Diabetes mellitus

Corticosteroid use

Preexisting edema in an

affected extremity

Animal bites

Infections that occur after a dog or cat bite are usually polymicrobial, involving a mixture of aerobes and anaerobes

P. multocida is the major pathogen

Organisms Most Frequently Isolated from Dog- and Cat-Bite Wounds

Aerobes	*Pasteurella multocida* *Corynebacterium* species *Staphylococcus* species *Streptococcus* species *Capnocytophaga canimorsus* (rare)
Anaerobes	*Bacteroides fragilis* *Veillonella parvula*

Wounds resulting from animal bites should immediately be washed with soap and water.

Infected wounds, wounds older than 12 hours, cat bites, and bites on the hand should be left open. In all cases of infection related to an animal bite, aerobic and anaerobic cultures should be obtained from the site of infection. Tetanus immune status should be determined, and immunization against tetanus should be provided when appropriate.

Wounds should be irrigated with povidone-iodine to reduce the transmission of rabies, and immunization against rabies should be provided.

Amoxicillin-clavulanate is the antibiotic of choice because of its broad spectrum of activity against common pathogens Patients with established soft tissue infection should receive antibiotic therapy.

Infections secondary to P. multocida respond to oral treatment with penicillin V, amoxicillin, cefuroxime, or ciprofloxacin.

Human bites:

Soft tissue infections resulting from human bites are polymicrobial, involving a mixture of aerobes and anaerobes. In addition, the concentration of bacteria in the oral cavity is higher in humans than in animals. The anaerobic bacteria Bacteroides species are more common.The predominant aerobic organisms in human-bite infections are S. aureus, Staphylococcus epidermidis. Other pathogens may also be transmitted as a result of contact with blood or

saliva, including hepatitis B and C viruses, Mycobacterium tuberculosis, and, possibly, HIV.

Aerobic and anaerobic cultures are obtained. Devitalized tissue should be debrided, and the wound should be left open, whether infected or not.

Antimicrobial therapy is indicated for all such injuries.Tetanus immunization status should be determined, and tetanus toxoid, tetanus immunoglobin, or both should be administered as indicated.

Patients with an uncomplicated human bite to the hand should receive a broad-spectrum oral antimicrobial agent, such as amoxicillin-clavulanate (or doxycycline if they are allergic to penicillins). Patients with systemic manifestations of infection -I.V. treatment include cefoxitin, cefotetan, and piperacillin-tazobactam.

OBSERVATION & RESULTS

ETIOLOGICAL FACTOR:

SPONTANEOUS-34%

TRAUMATIC-66% (TRIVIAL-99%, RTA-1%)

ONSET OF THE DISEASE

IT IS SEEN THAT 66 PERCENT OF THE INFECTIONS WHERE

FOLLOWIING TRAUMATIC ONSET.

34 PERCENT OF THE INFCTIONS WHERE SPONTANEOUS ONS

AGE DISTRIBUTION;

AGE	% OF INCIDENCE
0-10	8
10-20	6
20-30	10
30-40	6
40-50	**40**
50-60	16
60-70	12
70-80	2

AGE DISTRIBUTION;

THE INCIDENCE OS INFECTION IS HIGHEST AMONG THE AGE GROUP OF 40 TO 50 YEARS.

LEAST AMONG THE AGE GROUP IS 70 TO 80 YEARS.

IT IS 6 PERCENT AMONG THE AGE GROUP OF 10 TO 20 YEARS AND 30 TO 40 YEARS.

IT IS 8 PERCENT AMONG THE AGE GROUP OF LESS THAN 10 .

IT IS 10 PERCENT AMONG 20 TO 30 YEARS , 12 PERCENT AMONG THE 60 TO 70 YEARS AND 16 PERCENT AMONG THE 50 TO 60 YEARS OF AGE.

DEPENDING ON THE SITE

SITE	% OF DISTRIBUTION
HEAD & NECK	10%
UPPER LIMB	16%
CHEST WALL PARIETAL WALL	6%
EXTERNAL GENITALIA	28%
LOWER LIMB	40%

COMMONEST SITE

THE LOWER LIMB BEING THE MOST COMMON SITE OF INFECTION WHICH CONTRIBUTES TO 40 PERCENTAGE OF INFECTONS .

THEN COMES IN ORDER IS THE EXTERNAL GENITALIA WHICH CONTRIBUTES TO 28 PERCENTAGE , FOLLOWED BY UPPER LIMB OF 16 PERCENTAGE , THEN TH EHEAD AND NECK OF 10 PERCENTAGE , AN DTHE LEAST BEING TH ECHEST WALL AND THE PARIETES OF 6 PERCENTAGE.

MODE OF PRESENTATON:

ABSCESS AND CELLULITIS FORMS 40 PERCENTAGE OF THE INFECTONS

THEN FOLLOWS THE FOURNIERS GANGRENE OF 8 PERCENTAGE.

NECROTISING FASCITIS, THE ULCERS AND THE GANGRENE TOES FORMS THE 4 PERCENT OF THE REST OF INFECTIONS .

TYPES OF PRESENTATON

ABSCESS	40%
CELLULITIS	40%
NECROTISING FASCITIS	4%
GANGRENE	
TOES	4%
FOURNIERS GANGRENE	8%
ULCERS	4%

MOST COMMON ORGANISMS:

ORGANISMS	% OF INFECTIONS
STAPHYLOCOCCUS	22%
E.COLI	18%
PSEUDOMONAS	18%
PROTEUS	6%
STREPTOCOCCUS	6%
ENTEROBACTER	4%
NO GROWTH	26%

THE PREVELANCE OF INFECTION

CULTURES WHERE POSITIVE ONLY IN 74PERCENT OF INFECTIONS .

AMONG THE CULTURES GROWN IN STAPHYLOCOCCUS BEING THE MOST COMMON ORGANISM .

ESCHERICHEA COLI AND PSEUDOMONAS FORMS 18 PERCENTAGE OF INFECTIONS.

PROTEUS AND STREPTOCOCCUS FORMSTHE 6 PECENTAGE OF INFECTIONS.

THE REST 4 PERCENT OF INFECTIONS ARE FROM ENTEROBACTER GROWTH.

OTHER INFLUENCING FACTORS:

HB%--	<10GMS-	34%
DELAYED PRESENTATION-(>7DAYS)		34%
IMMUNO DEFICIENCY- (RVT, TB, MALNUTR.)		10%
NEURO VASCULAR DEFICIT		6%

THE OTHER FACTORS WHICH MAY BE A INFLUENCING IN THEIR ETIOLLOGY MAY BE A DELAY IN THEIR PRESENTATION AFTER THEIR INITIATING FACTOR. THE DELAYED PRESENTATION OF MORE THAN SEVEN DAYS WAS TAKEN IN TO ACCOUNT. IN OUR STUDY ABOUT THIRTY FOUR PERCENTAGE OF PATIENTS HAD A DELAYED PRESENTATION.

THIRTY FOUR PERCENT OF THE PATIENTS HAD A HEAMOGLOBIN OF LESS THAN TEN GRAMS PERCENT.

TEN PERCENT OF THE PATIENTS HAD SOMEFORM OF IMMUNODEFICIENCY AND SIX PERCENT OF THE PATIENTS HAD NEUROVASCULAR DEFICIENCY.

CONCLUSION OF THE STUDY:

1, IT IS SHOWN THAT STAPHYLOCOCCUS BEING THE MOST COMMON ORGANISM AS A CAUSE OF INFECTON.EVEN THOUGH CULTURES WHERE POSITIVE IN SEVENTY FOUR PERCENTAGE OF THE CASES ,SKIN COMMENSALS BEING THE MOST COMMON GROWTH .

2, THE CULTURES WERE NEGATIVE IN TWENTY SIX PERCENT OF THE PATIENTS AND ALL OF THEM GOT PRIMARY TREATMENT IN SOME OUTSIDE HOSPITAL BEFORE COMING TO US.

3, TRAUMA MAY BE THE INITIATING FACTOR FOR THESE SOFT TISSUE INFECTIONS AS IN OUR STUDY 66% OF THE PATIENTS HAS POSITIVE HISTORY OF TRAUMATIC ONSET.

4, ABSCESS AND CELLULITIS FORMS THE MAJORITY OF THE INFECTIONS.

5, THE COMMONEST SITE BEING THE LOWER LIMB AND THE EXTERNAL GENITALIA IN OUR STUDY.

6, THE AGE GROUP OF PRESENTATION WHICH IS MOST COMMON IS 40 TO 50 YEARS.

7, THE GENERAL CONDITION OF THE PATIENT LIKE THEIR HEAMOGLOBIN LEVEL AND IMMUUNODEFICIENCY STATUS SHOWED THAT THIRTY FOUR OF THE PATIENTS HAD HAEMOGLOBIN OF LESS THAN TEN PERCENT .

THIRTY FOUR PERCENT HAD A DELAYED PRESENTATION OF MORE THAN SEVEN DAYS.

8, IMMUNNODEFEICIENCY OF TEN PERCENT AND NEUROVASCULAR DEFICIENCY OF SIX PERCENT WHERE FOUND IN THE STUDY WHICH MAY BE THE INFLUENCING FACTOR FOR THESE PRESENTATION .

9, DEATH OF TWO PERCENTAGE AND COMPLICATIONS LIKE ORCHIDECTOMY OCCURRED IN SIX PERCENT OF THE PATIENTS.

PROFOMA:

NAME AGE SEX

IP NO UNIT WARD

OCCUPATION

H/O-

SWELLING SITE DURAT PAIN

@ ULCER @DILATED VEINS @ARTERIAL ULCER

@PIGMENTATION @DEFORMITY
@NEUROPATHY (NON DM)

@GANGRENE

PAST H/O-

HT TB IHD ALLERGEY

PERSONAL H/O-

SMOKING ALCOHOL BEETELNUT

FAMILY H/O-

EXAMINATION-

FEVER ANAEMIA JAUNDICE LYMPHADENOPATHY

LOCAL EXAM;

INSPECTION

SITE SIZE SURFACE EXTENT

ASSOCIATED-

PALPATION

TEMP TENDER EXTENT

DIAGNOSIS;

INVESTIGATIONS-

URINE R/E ALB- SUG- DEP-

BLOOD- HB%- TC- DC- ESR-

BT- CT-

STS/RVT-

PUS C/S-

BLOOD C/S-

X-RAY-

TREATMENT;

CONSERVATIVE-

SURGICAL- FASCIOTOMY

AMPUTATIONS-

HOSPITAL STAY-

FOLLOW UP-